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Guillain-Barre syndrome: a case report

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Guillain-Barre syndrome (GBS) is a complicated degenerative neurological disorder which can be acute or chronic in nature. It is an acquired condition which is characterized by progressive, symmetrical, proximal and distal tingling and weakness. Muscle stretch reflexes are decreased to absent and loss of sensation is common. Etiology remains unclear but pathophysiology includes demyelination of spinal nerve roots. Death is rare. Early diagnosis and prompt referral should occur in severe cases due to the incidence of potential ventilatory failure and cardiovascular instability in some patients. The case of a 37-year-old male presenting to a chiropractic office is described. The importance of a correct diagnosis by the chiropractor and the subsequent management is reviewed.
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KEY WORDS: polyradiculoneuritis, Guillain-Barre syndrome, extremity weakness, peripheral nerves, chiropractic.

Le syndrome de Guillain-Barré (SGB) est une manifestation complexe de dégénérescence neurologique de nature aiguë ou chronique. C'est un état acquis caractérisé par une faiblesse et des fourmillements progressifs, symétriques, localisés et distals. Les réflexes musculaires sont diminués ou absents et la perte de sensations est fréquente. L'étiologie demeure incertaine mais la physiopathologie comprend la démyélinisation des terminaisons nerveuses de la colonne. Ce syndrome est rarement mortel. Dans les cas graves, un diagnostic précoce et l'intervention rapide d'un spécialiste sont nécessaires en raison de la fréquence chez certains patients d'insuffisance ventilatoire et d'instabilité cardio-vasculaire possibles. On décrit le cas d'un patient de 37 ans se présentant au cabinet d'un chiropraticien. L'importance d'un diagnostic juste et d'un traitement adéquat de la part du chiropraticien sont examinés.
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MOTS CLÉS : polyradiculonévrite, syndrome de Guillain-Barré, faiblesse des extrémités, nerfs périphériques, chiropraxie.

Introduction

Many patients visit chiropractors because of neuro-musculo-skeletal complaints, usually those that involve the neck and lower back.[1,2,3] Some will visit chiropractors because of complaints with their extremities.

Peripheral neuropathy is a commonly encountered condition in clinical practice. Diabetes mellitus and excessive alcohol use coupled with a poor diet are the most common conditions found in patients with peripheral neuropathy. Multiple sclerosis should also be considered in patients who present with weakness and numbness in their extremities.[4,5] The most common cause of acute muscle weakness associated with peripheral neuropathy in adults is Guillain-Barre syndrome.[6]

This paper will discuss the causes, history, incidence and pathology of Guillain-Barre syndrome. The case of a 37-year-old male is also presented.

Case report

A 37-year-old male presented to a chiropractic office complaining about recent onset of tingling and numbness in both hands. He also complained about his feet which he described as "feeling sleepy." His symptoms began slowly following an upper respiratory tract infection that he had about 14 days prior. No history of trauma was reported nor had he ever complained of the current symptoms. He had a past history of mechanical back pain and received treatment including spinal manipulative therapy which gave complete relief.

Examination of this man revealed a healthy person in otherwise good condition. Generalized weakness of the lower extremities was noted when testing the quadriceps, hamstrings and the gastrocnemius-soleus muscle groups. Sensory loss was variable in both his hands and feet. Deep tendon reflexes of

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his upper and lower extremities were 1+. A general physical examination gave no other orthopedic, neurological, or organic findings. Respiratory and cardiac function was within normal limits.

The patient was referred to his physician for lab work. Blood tests were ordered and the findings included an increase in the white blood cell count. The conclusion drawn from this was that this patient had recently suffered from some type of infection which correlated well with his history of an upper respiratory tract infection.

A diagnosis of Guillain-Barre syndrome was made after utilizing the criteria that strongly support the diagnosis of Guillain Barre syndrome. Features required to rule out diagnoses other than Guillain Barre syndrome would include: no history of hexacarbon abuse, no evidence of porphyria, no history or culture evidence of diptheria, no history or evidence of lead intoxication, symptoms not purely sensory, no evidence of poliomyelitis, botulism, toxic neuropathy, or tick paralysis.[6] Therapeutic management of this included rest, nutritional support, patient education and periodic assessment of motor, respiratory, and cardiac function. Chiropractic care, which included spinal manipulative therapy to areas of subjective complaint was palliative.

No medical management was given by his physician. He was instructed to keep off of ladders for fear of falling and to abstain from heavy physical endeavors including sports.

This patient slowly improved so that he could return to work as an electrician. Five months later his sensory deficit was near normal and his leg strength had improved. He admitted that he is steadily returning to what he considers is "his old self".

Discussion

Guillain-Barre syndrome (GBS) is also known as polyradiculoneuritis, and chronic inflammatory demyelinating polyradiculopathy (CIDP).[7] In North America it has an incidence of 1–7 per 100,000 persons and as such is classified as one of the more common neurological conditions. It is the most common cause of acute weakness in patients under 40 years of age.[4] Diagnosis is difficult due to the lack of any test that is pathognomonic of this condition. It is a complicated disorder which can be chronic or acute in nature. It's etiology is unclear although it has been associated with both cell – and humoral – mediated autoimmune mechanisms.[8]

Guillain-Barre syndrome represents an important acquired condition that is acutely evolving, is immune mediated, and an inflammatory disorder of the peripheral nervous system. It progresses leading to demyelination and axonal loss. Clinical hallmarks are symmetrical flaccid muscle paresis and areflexia in the presence of an increased cerebrospinal fluid protein content, and electrophysiologic studies demonstrating evolving demyelination.[9] Causes such as viral infection,[10] surgery,[11,12,13] blood transfusion,[14] and mycoplasma infection,[15] have been implicated as a prodrome to this syndrome in many cases. The diagnosis of Guillain-Barre syndrome can be supported by the following clinical features as listed in Table 1.

Most patients develop a weakness which tends to begin in the lower extremities due to demyelination of the peripheral nerves resulting in ascending paralysis and also a loss of cranial nerve function.[15] Manifestations may be acute or chronic, and temporary or permanent, depending upon the degree of neuronal destruction.[8] Muscle stretch reflexes are depressed in most patients and the sensory loss is variable. Difficulty with walking, running, climbing stairs, and getting up from a chair are usual early complaints. This weakness is usually symmetric and can also involve the upper extremities.[7]

Of importance is the gamut of conditions that should be differentiated. Hexacarbon abuse, as in glue sniffing, can simulate Guillain-Barre syndrome. Diptheria presents similar findings in its later stages, therefore a recent infection of

Table 1
Clinical features that strongly support the diagnosis of Guillain-Barre syndrome

Progression over days to a few weeks
Relatively symmetrical
Mild sensory signs or symptoms
Cranial nerve involvement
Onset of recovery 2-4 weeks after halt of progression
Autonomic dysfunction
Initial absence of fever
Elevated CSF protein after 1 week of symptoms
Abnormal results of electrodiagnostic studies, with slowed conduction or prolonged F waves.

diptheria must be taken into consideration.[16] Others that should be considered are poliomyelitis, any evidence of porphyria, botulism, lead or arsenic intoxication, tick paralysis as in Lyme disease, and acute toxic neuropathies (from organophosphorus compounds). It is therefore important to accurately and thoroughly evaluate any cause of acute weakness.[17]

An objective index for diagnosis can be made utilizing motor evoked potentials (MEP). Cui found in 27 patients with GBS that the findings of MEP were consistent with pathological changes of demyelination and clinical distribution of muscle weakness. A good correlation between abnormal MEP and degree of muscle weakness ($p < 0.01$) was noted. He also found that MEP can be used for differential diagnosis as well as followup and effect of therapy.[18] MEP is painless and can be done easily and repeated.

The pathogenesis of Guillain-Barre syndrome shows edematous changes proximal to the spinal nerve root at the junction of the anterior and posterior roots. The myelin sheaths soon become irregular at about the third day. Lymphocytes appear about the ninth day and phagocytosis on the eleventh day. These findings were found in severe cases of this syndrome that led to death.[19]

Researchers believe that the myelin destruction is limited to those areas of nerve trunks with intense inflammation suggesting that the inflammatory cells have a direct effect initiating the demyelination. The demyelination occurs primarily in areas infiltrated with inflammatory cells. Degeneration of the basement membrane of the Schwann cell results.[8] This is due to macrophages in the presence of lymphocytes and not lymphocytes by themselves.[20] This breakdown, which is unclear is associated with an autoimmune attack on a component of peripheral myelin.[5,8] This attack is also mediated by T cells.[5]

Treatment

Treatment for patients with Guillain-Barre syndrome depends on whether they have mildly acute, severely acute or chronic involvement. The incidence of death in one study was 1.5% to 8% of patients.[21] Another study listed the death rate at 4%.[23] Other common complications include ventilatory failure and cardiovascular instability for which intensive care support should be utilized. Ventilatory failure is caused by involvement of airway and respiratory muscles, particularly the diaphragm. Cardiovascular instability is due to involvement of the autonomic nervous system and results in labile blood pressure, cardiac arrhythmias, and hypovolemia. Compressive neuropathies occur in patients with protracted weakness and are an important cause of residual neurological deficits.

The use of corticosteroids demonstrated no benefit.[23] The only well-investigated efficacious immunomodulatory therapy is plasmapheresis.[9] Plasmapheresis has been shown to decrease ventilator dependence in severe Guillain-Barre syndrome. No irreversible complications of plasmapheresis were observed. In all cases of this condition special emphasis should be given to psychologic support and management of pain.[9] A persistent residual paresis occurs in severe cases and a high majority of these patients ultimately have a good functional recovery and can recover completely at about 12 months after onset.[22]

Conclusion

Guillain-Barre syndrome is a neurological disorder resulting primarily in muscle paralysis that in most cases is symmetrical. Patients may have mild involvement or severe involvement which may in a small percentage lead to death. Patients may present to a chiropractic office with symptoms of polyradiculoneuralgia which may at first be interpreted as a radicular pain pattern of spinal origin. It is extremely important to identify and urgently refer, potential severe cases in order to have the appropriate investigations (i.e. electrodiagnostic studies, spinal tap) instituted and have the appropriate care administered. Differential diagnosis is of utmost importance.

References

1 Till G, Mior SA, McGregor M. A study of chiropractic patient population in Saskatoon. Proceedings of the World Federation of Chiropractic: Toronto 1991:28.
2 Shekelle PG, Brook RH. A coummunity based study of the use of chiropractic services. Am J Public Health 1991; 81:439–442.
3 Oswald IH, Coulter I. Chiropractors do they help? Don Mills, Fitzhenry and Whiteside Limited, 1990:135–138.
4 Kraft GH, Freal MS, Coryell JK. Disability, disease duration and rehabilitation service needs in multiple sclerosis: patient perspectives. Arch Phys Med Rehab, 1986; 67:164.
5 Hartung HP. Immune-mediated demyelination (comment). Annals Neurology 1993; 33(6):583–587.
6 Miller RG. Guillain Barre syndrome. Postgraduate Medicine 1985; 77(7):57–64.
7 Mendell JR. Chronic inflammatory demyelinating polyradiculopathy. Annual Review Medicine 1993; 44:211-219.
8 Murray DP. Impaired mobility: Gullain-Barre syndrome. J Neuroscience Nursing 1993; 25(2):100–104.
9 Hund EF, Borel CO, Cornblath DR, Hanley DF, McKhann GM. Intensive management and treatment of severe Guillain-Barre syndrome. Critical Care Medicine 1993; 21(3):433–446.
10 Ormerod IE, Ceekerell OC. Guillain-Barre syndrome after herpes zoster infection, a report of 2 cases. European Neurology 1993; 33(2):136–318.
11 Stambough JL, Quinlan JG, Swanson JD. Guillain-Barre syndrome following spinal fusion for adult scoliosis. Spine 1990; 15(1):45–46.
12 Ennis JH, Bednar DA. Lumbosacral fusion in a patient with recurring Guillain-Barre syndrome and acute brachial neuritis. J Spine Disord 1992; 5(2):217–218.
13 Ginn D. Guillain-Barre syndrome. An uncommon but severe illness. Postgrad Med 1991; 90(5):145–416, 153–156.
14 Merelli E, Sola P, LaSpina I, Orlando A, Milanti G. Guillain-Barre polyradiculoneuritis after blood transfusion. Ital J Neuro Sci 1991; 12(3):313–315.
15 Nadkarni N, Lisak RP. Guillain-Barre syndrome (GBS) with bilateral optic neuritis and central white matter disease. Neurology 1993; 43(4):842–843.
16 Macleod J. Davidson's Principles and Practice of Medicine. Eleventh Edition, New York: Churchill Livingstone 1975:65–68.

17 Felt H, Tindall RS, Glasberg M. Sources of error in the diagnosis of Guillain-Barre syndrome. Muscle Nerve 1982; 5(2):111–117.
18 Cui LY. Magnetic stimulation motor evoked potential in patients with Guillain-Barre syndrome (Chinese). Chung-Mua I Msueh Tsa Chih 1992; 72(10);600–603, 639.
19 Haymaker W, Kernohan JW. Landry-Guillain-Barre syndrome: clinicopathologic report of 50 fatal cases and a critique of the literature. Medicine (Baltimore) 1949; 28:59–141.
20 Prineas JW. Pathology of the Guillain-Barre syndrome. Ann Neurol 1981; 9(suppl):6–19.
21 Ropper AH. Management of Guillain-Barre syndrome. In: Ropper AH, Kennedy SK, Zervos NT, Ed. Neurological and neurosurgical intensive care. Baltimore: University Park Press, 1983:163.
22 Smith GD, Hughes RA. Plasma exchange treatment and prognosis of Guillain-Barre syndrome. Quarterly J Medicine 1992; 85(306):751–760.
23 Hughes RA, Newsom-Davis JM, Perkin GD, et al. Controlled trial of prednisolone in acute polyneuropathy. Lancet 1978; 2(8093):750–753.